it was given intravenously in doses of 0.02 to 0.03 mg. a blood-pressure rise was always established. There were great variations in the reactions from subcutaneous and intramuscular injection of adrenalin, but in no case did he fail to get a rise in blood-pressure from intravenous administration, regardless of whether or not reactions were obtained by the other modes of administration.

Salvarsan in Malaria.—GLASER (*Deutsch. med. Wchnschr.*, 1921, xlvii, 867) reports the case of a syphilitic, aged twenty-eight years, who knew nothing about his latent malarial infection and who went through a course of salvarsan which provoked such a serious attack of "tropical malaria" that within fourteen days he was dead. In war veterans with unexplained postsalvarsan febrile symptoms one should always think of malaria. In ordinary forms of malaria salvarsan is curative, but in "tropical malaria" it does not sterilize the blood and in doses of 0.4 to 0.6 gm. stimulates the tropical malaria parasite. It seems to cause a sweeping of the organism into the blood stream.

PEDIATRICS

UNDER THE CHARGE OF

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Experimental Studies on Hydrocephalus.—NANAGAS (*Bull. Johns Hopkins Hosp.*, December, 1921) says that the pressure of cerebrospinal fluid in kittens in which an internal hydrocephalus had been experimentally produced was considerably higher than that of normal kittens. An average difference of 50 per cent was found in his series of observations. The maximal recorded cerebrospinal-fluid pressure obtained in this series was 171 mm.; the average of all cases was 126 mm., and the lowest was 100 mm. The average pressure of normal kittens was 76 mm.; the highest was 102 mm., and the minimal was 66 mm. Intravenous injections of a strongly hypertonic solution of sodium chloride in hydrocephalic animals produced a brief initial rise in the pressure of the intraventricular pressure of the cerebrospinal fluid, which was followed immediately by a marked depression. This decrease in pressure at times produced negative values. This phenomenon is probably to be explained by the apparently rapid absorption of the cerebrospinal fluid from the dilated cerebral ventricles. Intravenous injections of hypotonic solutions, such as distilled water, in hydrocephalic kittens was invariably followed by a marked and sustained increase in the pressure of the cerebrospinal fluid. This result was probably due to a rapid elaboration of fluid by the choroid plexus, or to an increased transudation of fluid through the ventricular walls. Intraventricular absorption of cerebrospinal fluid took place in these hydrocephalic kittens, and the pathway of absorption was through the ependyma into the underlying capillary network. This absorption of cerebrospinal fluid was hastened by the intravenous injection of strongly hypertonic solutions. Absorption of the cerebrospinal fluid was similarly found taking place through the ventricular ependyma in normal kittens. The pathway of escape was the same as described above. The process of absorption was slow in the normal animals, but it was hastened by the administration of hypertonic solutions. The physiological significance of this intraventricular absorption in the normal animals is probably minimal. The choroid plexus took absolutely no part in the intraventricular absorption of the cerebrospinal fluid.

The Nature of the Plantar Reflex in Early Life and the Causes of its Variation.—Feldman (*Am. Jour. Dis. Children*, January, 1922) studied 500 subjects from birth to seven years of age, most of whom were younger than four years. He obtained plantar response in 426 cases. He found that the prevailing plantar response in early life is plantar flexion of the big toe, although when a dorsiflexion of the toe occurred it was not of the same significance as the similar response in the adult. The pyramidal tracts are sufficiently developed at birth at full term to give an adult normal type of plantar reflex even intra-uterinely. Owing to easily aroused circulatory disturbances in early life the consequent changes in the circulation in the region of the cord are sufficient to compress the incompletely myelinated pyramidal tracts to evoke a Babinski phenomenon either unilaterally or bilaterally. In premature infants the response is nearly always of the Babinski type, up to five or six weeks of postnatal life, because of the almost total absence of myelization of the pyramidal tracts. Malnutrition, as judged by defective weight and length, go hand-in-hand up to about five or six weeks. The Babinski sign seen in badly developed infants up to that age is due to prematurity rather than to the malnutrition. Bilateral plantar flexion is at all ages as common in girls as in boys, but bilateral dorsiflexion is at all age periods in infancy more common in girls. Breast-feeding during the first four weeks of life probably tends to diminish the incidence of the Babinski phenomenon. This may be due to the greater percentage of lecithin and lactose in human milk, which helps the more rapid myelization of the pyramidal tracts. After the first month or so breast-feeding has no advantage in this respect over bottle-feeding. Toxic influences, either from the bowel or other causes, do not affect the conductivity of impulses along the fibers of the pyramidal tracts. Bilateral dorsiflexion is slightly more common in dolichocephalic than in brachiocephalic infants, possibly because inhibitory control is less powerful in the former than in the latter. Rickets does not favor the occurrence of the Babinski reflex. The age at which the sign vanishes has no relation to the age at which the child begins to walk. In the majority of the cases of very young infants who could not even sit up the Babinski sign was absent, and in the large number of cases of the children who could walk the Babinski sign was present. As the peripheral nerves are imperfectly myelinated at birth a possible explanation of the Babinski phenomenom in certain infants is the more imperfect development of the lower motor neuron supplying the flexors of the toes, and better development of the neuron